A nutritional primer for anesthetists

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As the author states, incorporation of a nutritional perspective into the care of the anesthetized patient may improve the outcome. The following article explores those aspects of nutrition of particular relevance to the clinical anesthetist.

The broad, interdisciplinary science of nutrition is becoming increasingly complex; only relatively recently has medical science become avidly interested in the practical, clinical application of nutrition. As the intimate relationship between nutrition and various physiological states becomes increasingly apparent, it is incumbent upon anesthetists to incorporate a nutritional perspective into anesthetic care.

Data exists indicating that the nutritional state has a direct influence on the operational level of the immune system and other organ systems that are related to survival. A vicious cycle exists in that infection occurs more often and with greater severity in the poorly nourished, and that the infectious state contributes to a decline in nutritional status. Nutritional deficiencies of fat and protein are also common in pediatric medical and surgical intensive care units.

All three substrates may participate as energy sources via various pathways. (Figure 1.) Carbohydrates utilize both anaerobic and aerobic routes (glycogen breaks down anaerobically to lactic acid; glycogen breaks down aerobically to pyruvic acid, which is then converted to acetyl CoA and enters the Krebs cycle). Protein breakdown provides amino acids (ketogenic or glucogenic) for utilization. Neutral fat undergoes hydrolysis to glycerol and fatty acids with eventual conversion to acetyl CoA and entry into the Krebs cycle. With prolonged starvation, protein catabolism becomes a highly important source of energy.

In the healthy individual, protein is not stored for energy purposes. Caloric reserves generally are found in triglycerides stored in adipose tissue (approximately 130,000 kcal even in the nonobese), with much smaller amounts available from blood glucose and liver/muscle glycogen (approximately 1000 kcal). Protein in the body (16,000-24,000 kcal) is utilized very specifically, and depletion of body protein as an energy substrate will eventually lead to the impairment of the organism. Duration of the substrate reserves under basal conditions are: 20-25 days (fat), 6-12 hours (carbohydrates), and 10-12 days (protein).

"Nitrogen pool," an all-encompassing phrase,
refers to all the amino acids and other simple nitrogenous substances in the body; it is maintained by a complex set of pathways providing for degradation and resynthesis of amino acids from exogenous and endogenous sources. Nitrogen balance is said to occur when as much nitrogen is consumed as is excreted. A catabolic state results in the marked loss of nitrogen and an upset in this delicate balance. It is a dictum that any major surgical procedure results in six weeks of catabolism.

The respiratory quotient (RQ) is the ratio of carbon dioxide output to oxygen consumed. When a molecule of glucose is oxidized, the number of molecules of carbon dioxide released is exactly equal to the number of oxygen molecules utilized in the oxidative proceedings; the RQ here is 1.00.

The oxidation of fat yields an RQ of 0.71; with proteins the RQ is 0.83. High glucose intake associated with administration of total parenteral nutrition tends to increase carbon dioxide production.

It has been suggested that such carbon dioxide production might precipitate respiratory complications in the patient with compromised pulmonary function. In acutely ill and nutritionally depleted patients the use of fat emulsions in moderate amounts can provide for a significant reduction in carbon dioxide production and therefore in ventilatory requirements. Manipulation of the metabolic substrate regimen may confer critical benefit to the patient with compromised pulmonary reserves.

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**Figure 1**

Metabolic integration of carbohydrates, fat and protein

![Metabolic diagram](https://example.com/metabolic_diagram.png)


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A significant portion of the basal metabolic expenditure (approximately 20%) is utilized to fuel the central nervous system. It was thought at one time that the only substrate capable of meeting the energy requirements of the central nervous system was glucose; however, it is now known that the central nervous system (as well as the myocardium and liver) can utilize ketone bodies and free fatty acids. The influence of anesthetics on myocardial and hepatic systems is coming to light: there is now data available to suggest that a course of starvation can attenuate rather than potentiate the anesthetic depression of myocardial and hepatic performance in animals.\(^2\) Anesthetics, then, do influence substrate utilization and therefore metabolic function.

Starvation blunts the ventilatory responses to hypoxia and hypercarbia.\(^7\) Prolonged limitation of nutritional intake to a 5% dextrose solution can induce just such a state.\(^8\) This phenomenon can rapidly be reversed by a protein-rich meal.\(^9\) Similarly, total parenteral nutrition will augment the ventilatory response to carbon dioxide which has been depressed in a semi-starvation (prolonged 5% dextrose in water) state.

The exact amount of protein loss required to cause death is not known. In starvation, skeletal and cardiac muscle is wasted. Visceral proteins are depleted and the immune system undergoes deterioration. Morbidity and mortality associated with surgery on the malnourished are high, a fact appreciated by trauma surgeons.\(^10\) Pathology results from disruption of the synthesis of enzymes and protein molecules intracellularly.

Cell-mediated immunity is important in the defense against infection. Morbidity and mortality increase as this system is obtruded. The surgical patient whose immune system is depressed is particularly at risk.\(^11\) Short-term nutritional repletion is a proven method of dealing with the problem.\(^12\)

Glucose intolerance is a frequently observed phenomenon in injured and infected patients, and may act as a significant obstacle to adequate nutritional support.\(^13\) Altered patterns of fuel utilization in this category of patients are poorly understood but may be related to the altered relationships between free fatty acids and cellular metabolism.

**Assessing the patient**

Various means of nutritional assessments are available, some more complex than others. A busy clinician may not view with favor the suggestion to determine the patient's nutritional status during the preoperative assessment. However, a “streamlined” approach based upon sound nutritional principles will do much to uncover nutritionally vulnerable patients and may improve outcomes.

An excellent indicator of protein-calorie malnutrition is a recent weight loss of 10% or more. The time frame involved is of critical importance. If occurring over a two week period, without accompanying infection, such a loss is probably primarily of fluid. However, such a loss occurring over a one to three month period is suggestive of loss of lean and adipose mass and may indicate the development of a protein-calorie depletion state (unless the patient is involved in a sound weight reduction program).

Starvation by itself is not a life-threatening situation until a 40% weight loss has occurred.\(^24\) However, if illness or injury is superimposed on a starvation state, high death rates occur after a 25% weight loss.\(^25\) The mechanisms responsible are well understood.\(^26,27\) The normal response to a starvation state is conservation: reduction in metabolic rate and sparing of lean body mass by increased central nervous system utilization of ketones. In the stress state combined with starvation, the metabolic rate is not decreased, gluconeogenesis continues and catabolism of protein stores is perpetuated.

The role of the clinical nutritionist as a member of the patient care team is well established. This professional's assistance should be sought when appropriate.\(^28\) Anthropometric measurements should be undertaken by those familiar with the techniques. These techniques are described in detail in literature.\(^30\)

Indicators of nutritional status include calculation of caloric expenditure by measurement of oxygen consumption; calculation of nitrogen balance using a 24-hour urine test with application of the Micro-Kjeldahl methodology;\(^31\) determination of visceral protein status (serum albumin and serum transferrin studies); and immune competence testing.

When obtaining the patient's history, the anesthetist should be alert to signs that might signal possible malnutrition: alcohol/drug dependency, physical disability, preoccupation with food fads, altered lifestyle, socioeconomic disability, personality changes, and extremes of age. A history of prolonged diarrhea, chronic nausea or vomiting, anorexia or lack of energy also deserve further investigation to determine if a nutritional deficiency exists. The anesthetist should determine the patient's estimated height and weight, any recent weight change, current diet and past dietary repl-
restrictions. The patient's chart will furnish information on specific problems, diagnoses, presence of fever, blood pressure, diet order, and laboratory reports.

**Total parenteral nutritional update**

Ideally, caloric protein and electrolyte administration should be individualized. Adults require approximately 40 nonprotein kcal/kg/day. The average adult requires approximately 11-15 gm of nitrogen each day (0.2 gm/kg/day). Hospitalized patients are frequently provided with more protein to offset a hypercatabolic state. Six and one-quarter gm of protein, equivalent to 30 gm of muscle, contain about one gm of nitrogen. Thus, the protein requirement for an average adult patient is 70-90 gm/day. This can be met with 1500 cc of a 5.5% amino acid vehicle. The optimum ratio of nonprotein calories to gm of nitrogen is 140:1. Fat provisions should not exceed 3-4 gm/kg/day. Electrolyte requirements have been thoroughly described in literature. These include potassium needs of 48-180 mEq/day and sodium needs of 48-125 mEq/day.

Intralipid, the prototype fat emulsion, is made up of 10% soybean oil and egg phospholipid. It is isotonic and neutral, providing 1.1 kcal/cc, and can be given via a peripheral vein. A 20% emulsion exists, providing 2 kcal/cc; the advantage being the ability to reduce fluid volume in the hyperalimentation regimen.

A basic principle of hyperalimentation is that fat does not provide a significant nitrogen-sparing effect. Although fat provides 9 kcal/gm (or about twice that of protein or carbohydrate) it cannot be used as the sole caloric source. Dilute dextrose-amino acid solutions should be utilized concurrently with the fat emulsion. A minimum of 30% of the total substrate provided should be in the form of carbohydrates.

The intravenous route of nutrition is unphysiological. When isocaloric, isonitrogenous diets are fed patients both intravenously and orally, the orally fed group demonstrates superior results. Superior vena cava entry changes the normal portal venous blood flow to the liver and bypasses normal metabolic processes which occur in the gut. Intraportal administration is associated with extreme hazards (such as portal thrombosis) and will not likely supplant current methods.

One should be aware that it is not always absolutely essential to infuse hypertonic solutions via a central line to provide for essential nutrition. A practical guide is that the administration of more than 2,000 nonprotein calories per day via a peripheral vein is associated with an increased incidence of complications, such as thrombophlebitis and fluid overload.

Hazards associated with total parenteral nutrition (TPN) have led many to approach the topic with new perspective. Hyperosmolar, hyperglycemic, nonketotic coma is seen with relative frequency in hyperalimentation patients. Discontinuing hyperalimentation regimens inappropriately can precipitate fatal hypoglycemic episodes. Hypophosphatemia, metabolic acidosis, metabolic alkalosis, infection, and technical problems associated with centrally dwelling lines (such as hydrothorax, pneumothorax, and mediastinal hematoma) make TPN regimens hazardous.

Canadian workers have proposed that bronchial administration of isotonic glucose and insulin might maintain alveolar metabolism. Anaerobic utilization to maintain adenosine triphosphate levels has been demonstrated. Practical application might extend to the patient with sepsis, pulmonary trauma, aspiration, head injury, fat embolization and adult respiratory distress syndrome.

**Perioperative nutritional principles**

Ideally, a diet adequate in calories, high in carbohydrates and containing ample protein should be provided for at least 7-14 days prior to surgery. The extra carbohydrates will spare body proteins and the storage of glucose and glycogen will help to protect the liver and mitigate the occurrence of postoperative ketosis.

If fasting is initiated acutely and continued for a 24-hour period, a healthy adult (utilizing 1800 kcal/day) will burn 75 gm of protein and 160 gm of adipose-tissue triglycerides. The organ playing the paramount role is the liver, which will maintain glucose levels by the process of gluconeogenesis (converting glycerol, lactate and specific amino acids to glucose). The postoperative patient experiencing an uncomplicated course will require 75-125 gm of protein 2000-3500 kcal) in a 24-hour period; in the event of a hypercatabolic state (in cases of multiple injuries or burns), the same patient will require 100-300 gm of protein (3800-5000 kcal).

The well-nourished patient presenting for elective surgery can easily handle a 2-3 day fast that might be associated with the surgical procedure. The more chronically ill or traumatized patient who may not be so well nourished may require nutritional interventions (nasogastric or parenteral feeding) immediately.

The anesthetist frequently sees patients being maintained on the proverbial 100 gm of glucose...
per day. This concept was based on early work by Gamble which has since been shown incorrect. The classic paper by Dudrick and associates demonstrated that intravenous delivery of nutrients to beagle puppies supported growth and development, and the era of nutritional support was upon us.

The ethanol-nutrition interaction deserves special mention. Effects occur at at least three levels:

1. Displacement of food from the diet and suppression of appetite.
2. Compromised function of liver, pancreas or bowel.
3. Metabolism of ethanol to acetaldehyde with secondary alterations of function.

The hepatic toxicity of alcohol is firmly established; hepatic steatosis and cirrhosis can ensue despite nutritional supplementation.

Although one gram of ethanol yields 7.1 calories, ethanol is not a caloric substitute. Protein synthesis is depressed in chronic alcoholics. Autonomic dysfunction, impaired gluconeogenesis and glycogen depletion have been related to ethanol intake. A whole variety of hematological, cardiovascular and neurological problems are experienced by the chronic abuser.

Patients with chronic renal disease are predisposed to malnutrition. Improved understanding of protein metabolism has improved clinical management. There are multiple special problems associated with TPN in this patient population.

The acutely ill patient with impaired renal function presents a particularly challenging problem. Mortality is especially high in this group. Dialysis exacerbates the catabolism of the illness.

Starvation states associated with specific disorders such as hepatic failure have implications for the anesthetic management of the patient. Plasma amino acid alterations influence central neurotransmitter substances and can influence consciousness. MAC and arousal from anesthesia may

<table>
<thead>
<tr>
<th>Class</th>
<th>Possible Interactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antacids</td>
<td>Sodium, potassium, calcium, calorie content. Excess magnesium may induce diarrhea.</td>
</tr>
<tr>
<td></td>
<td>Excess aluminum may induce constipation. Excess calcium may induce constipation.</td>
</tr>
<tr>
<td></td>
<td>Note administration time: may inhibit absorption of nutrients.</td>
</tr>
<tr>
<td>Laxatives</td>
<td>May promote fluid and electrolyte loss; sodium and calorie content.</td>
</tr>
<tr>
<td>Analgesics</td>
<td>Gastric irritants, possible bad taste. Narcotic analgesics: constipation; nausea and</td>
</tr>
<tr>
<td></td>
<td>vomiting; decrease in gastric motility.</td>
</tr>
<tr>
<td>Antidepressants-antisychotics</td>
<td>May induce increased blood glucose levels; possible induction of metallic taste;</td>
</tr>
<tr>
<td></td>
<td>stimulate appetite; possible weight gain.</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>May increase blood glucose levels; inhibit absorption of enzymes; increase turnover</td>
</tr>
<tr>
<td></td>
<td>of folic acid, vitamin D.</td>
</tr>
<tr>
<td>Antibiotics</td>
<td>Sodium, potassium content; possible bad taste; possible diarrhea, gastric upset.</td>
</tr>
<tr>
<td></td>
<td>Note administration time.</td>
</tr>
<tr>
<td>Cytotoxic drugs</td>
<td>Nausea and vomiting; diarrhea; constipation; stomatitis; glossitis.</td>
</tr>
<tr>
<td>Autonomic drugs</td>
<td>Affect carbohydrate metabolism; affect digestion; decrease bowel motility; nausea;</td>
</tr>
<tr>
<td></td>
<td>vomiting; epigastric distress.</td>
</tr>
<tr>
<td>Corticosteroids, other anti-inflamatory agents</td>
<td>Gastric upset; sodium and water retention; increase blood glucose levels.</td>
</tr>
<tr>
<td>Oral contraceptives</td>
<td>Increase blood glucose levels; decrease folate levels; salt and water retention.</td>
</tr>
<tr>
<td>Hypcholesteremic agents</td>
<td>Decrease fat absorption; bind bile salts.</td>
</tr>
</tbody>
</table>

be altered and will influence the appropriate selection of anesthetic technique.47

**Drug-nutrient interrelationships**

Since the reports in the early 1960s of dramatic food-drug interactions involving pressor-amine containing foods and monoamine oxidase inhibitors, efforts have been made to predict and thus prevent the occurrence of adverse interactions.48 The nutritional status of the patient modifies the action of a drug in the body.49,50 The amount of “free drug” available to receptors, as well as drug pharmacokinetics may be altered. Certain drugs may be metabolized faster when the individual is consuming a high protein/low carbohydrate diet.51 Special diets may lead to enhancement of hepatic enzymes.52,53

Monoamine oxidase inhibitor drugs elevate levels of norepinephrine and serotonin in the central nervous system and potentiate the cardiovascular effects of simple phenylethylamines such as tyramine.54 Foods producing hypertensive crisis in patients taking MAO inhibitor drugs include those that are high in tyramine or dopamine.

An example of a drug-nutrient interaction is that of alcohol and disulfiram. Disulfiram blocks the oxidation of alcohol at the acetaldehyde stage; accumulation of acetaldehyde produces unpleasant symptoms.55 Individuals taking diet aids containing phenylpropanolamine may experience serious central nervous system symptoms. Anxiety reactions, agitation, dizziness, hallucinations, tachyplea and tachycardia are all reported.56 Concomitant administration of a sympathomimetic drug may exacerbate the phenomenon.

**Conclusion**

Intensive, holistic care of patients who are to undergo anesthesia and surgery demands attention to nutritional considerations. State of the art management requires that anesthesia practitioners be cognizant of scientific principles regarding nutrition and apply these principles intelligently in clinical situations.

**REFERENCES**


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**Table II**

**Autonomic drug interactions**

<table>
<thead>
<tr>
<th>Class of autonomic drug</th>
<th>Examples</th>
<th>Interactions/side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathomimetic (adrenergic agents)</td>
<td>Anorexic drugs, Decongestants, Respiratory stimulants, Vasoactive drugs</td>
<td>Hyperglycemia, Decrease GI motility, Depress appetite, Increase free fatty acid level, Headache, Nausea and vomiting</td>
</tr>
<tr>
<td>Sympatholytic</td>
<td>Adrenergic blockers (i.e., Inderal®)</td>
<td>Hyperglycemia, Nausea and vomiting</td>
</tr>
<tr>
<td>Cholinergic (parasympathomimetic)</td>
<td>Ophthalmics (no systemic effects to note), Urecholine®—used postop to relax urinary bladder</td>
<td>Dry mouth, Increase gastric motility</td>
</tr>
<tr>
<td>Anticholinergic (biggest offender) (parasympatholytic)</td>
<td>Peptic ulcer drugs, preop cocktail (Atropine, Donnatal®, Cogentin®, Norflex®)</td>
<td>Dry mouth—decrease salivary secretion, Constipation, Nausea and vomiting, Possible interference with absorption of nutrients, Decrease gastric secretions</td>
</tr>
</tbody>
</table>